Modern Concepts of Cardiovascular Disease

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CORONARY THROMBOSIS

During the past twenty years coronary thrombosis has been recognized with increasing frequency and has rapidly supplanted the old diagnosis of "acute indigestion". The increased frequency of recognition is due in great part to accurate clinical observations and descriptions of the disease by American investigators.

This disease occurs most commonly in patients between the ages of fifty and seventy years and is from three to four times more frequent in males. It does occur in younger individuals and there are reported instances where patients in the thirties have been attacked.

Obstruction of the coronary artery usually results from one of the following causes: (1) From a thrombus originating in a coronary artery which previously shows evidence either of atheromatous degeneration or arteriosclerosis. This is by far the most common lesion present and roughly estimated can be demonstrated in approximately ninety per cent of the patients coming to necropsy. (2) As the result of narrowing of the ostium of the artery just as it leaves the aorta. This type of lesion is observed in individuals who have syphilitic aortitis, but very rarely indeed does the syphilitic process advance to such a degree that obstruction to the blood flow occurs. (3) From an embolus originating within the heart or its valves and lodging in the artery. This type of lesion rarely occurs in patients with bacterial endocarditis.

Thrombosis may occur very slowly or very suddenly. Should the occlusion take place gradually, there are no acute symptoms; the picture is one of a myocardium which slowly fatigues and the symptoms are those of a progressively increasing congestive heart failure. With sudden occlusion, the symptoms are acute, serious and dramatic. In the one instance there occurs a slow gradual degeneration of the heart muscle with an increasing fibrosis, stretching and dilatation; in the other, there is the formation of an acute infarct in the myocardium, the extent of which depends on the size of the vessel occluded and on the efficiency of the collateral circulation. Should a large artery suddenly become occluded, death occurs within a few minutes or several hours; should one of the smaller arteries become occluded, recovery usually takes place. While either the right or the left coronary artery may be occluded, the anterior descending branch of the left coronary is the one most frequently involved and as this artery supplies the apical portion of the left ventricle, the interventricular septum and the papillary muscle, this region of the heart is most commonly affected. The anaemic infarct which results from this lesion may undergo acute degenerative changes which will cause the heart muscle to so weaken that it ruptures, or with healing, there is fibrosis and the production of an aneurysm or there may form within the ventricle mural thrombi which can become dislodged and carried to other parts of the body. Most patients with coronary thrombosis have arteriosclerosis and hypertension although in a considerable number, hypertension has not existed; occasionally the arteriosclerosis is most extensive in the coronary arteries, the other arteries of the body showing very little evidence of the disease.

The attacks with which we are most familiar begin suddenly while the patient is at rest. They occur at times during sleep and occasionally after a full meal (in this respect differing from the onset of angina pectoris which follows exercise or emotional strain). The pain is sudden and usually is referred to the lower sternum or to the epigastrium. It increases in intensity and spreads to the upper sternum, to the precordium, to the left or right arm; it may be referred to the back, the neck or jaw, and very frequently may involve the entire chest. In some patients the pain is referred entirely to the abdomen, in this respect resembling that

occurring in acute pancreatitis, cholelithiasis or perforating peptic ulcer. The pain is described as burning, boring, cramping or vise-like in character; "it cuts off the breath" and makes breathing difficult. It lasts for hours at a time and is uninfluenced by nitrites. The victim is restless, tosses about in bed or is up walking about the room; he can't keep still; there is marked mental anguish; the skin is cold and clammy and sweat covers the brow. True dyspnoea does not occur early except in those patients who show marked heart failure, but difficulty in breathing is an outstanding complaint. In an attack death may occur suddenly, within a few minutes, or hours, or recovery may take place. Those patients who survive the attack exhibit a striking series of changes; the blood pressure, systolic and diastolic, falls, this change being observed most noticeably in those who have previously had hypertension. The heart rate increases and as a rule remains regular, but various types of irregularity may occur, such as paroxysmal auricular fibrillation, coupled beats, extra systoles, heart block or ventricular tachycardia. The first heart sound becomes muffled and impure. A pericardial friction rub, which is of short duration, can frequently be detected over the infarcted area. Fever occurs and lasts for several days gradually subsiding. A leucocytosis from ten to twenty thousand comes on within a few hours and persists for several days. Cough, dyspnoea, cyanosis, oedema of the lungs, enlargement of the liver may occur with progressive myocardial failure.

The electrocardiogram is of much value in establishing a diagnosis; in many cases typical and characteristic changes are observed, although it must be remembered that a few patients with coronary thrombosis may have a normal electrocardiogram.

The outcome in a given case is marked with the greatest uncertainty; it occasionally happens that a patient, who for days is progressing favorably, will die very suddenly, and another who seemingly has advanced congestive heart failure with marked cardiac irregularity, an almost imperceptible pulse, a blood pressure that can't be measured, will recover. Approximately forty per cent of patients die suddenly or within a few hours in the first attack; those who recover may live for one or more years in a moderate degree of comfort.

Treatment resolves itself into the care of the patient during the acute attack and, in those who survive, during convalescence. During the acute attack relief of pain can only be accomplished, and even then too slowly, by giving ½ grain of morphine hypodermically and repeating as necessary. Other substitutes for this drug are worthless and smaller doses do no good. The earlier the pain is relieved, the less the shock. For those patients showing circulatory collapse during the first few hours of the illness, intravenous injections of 40 to 50cc of a buffered fifty per cent solution of glucose are of value. Stimulation is also indicated, using such drugs as euphyllin, caffein sodium benzoate, digitalis or strophanthin intravenously; adrenalin subcutaneously may be of value in tiding the patient over a period of acute circulatory failure. Experience seems to show that so long as the heart can maintain a fairly efficient circulation, it is much better to do without stimulants, except drugs, such as euphyllin, which increase the coronary circulation. Should circulatory failure occur toward the end of the first week of the disease, it should be treated as at other times always, however, using, digitalis with caution, since in this condition it is possible that it might produce ventricular tachycardia and death. Quinidin is of value in treating those patients who develop ventricular tachycardia and is often life saving. It must be used with the greatest care and caution. At least six weeks of bed rest should be prescribed before the patient is allowed to be up; his future activities are to be determined by the condition of his myocardium, which even at best, must be considered as having been badly damaged.

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SELECTED ABSTRACT

McEachern, D., and Baker, Jr., B. M.: Auricular Fibrillation. Its Etiology, Age Incidence and Production by Digitalis Therapy. Amer. Jour. Med. Sci. 183: 35: (Jan.) 1932.

McEachern and Baker report a study of 575 cases of auricular fibrillation seen at the Johns Hopkins Hospital. Thirty-four per cent of the patients had rheumatic heart disease, 31% arteriosclerosis with relatively low blood pressure, 17% hypertension (systolic pressure over 150 mm. of mercury) and 7.5% hyperthyroidism. The authors call attention to the rarity of this arrhythmia in syphilitic heart disease, and in juvenile cardiacs under 10 years of age. Several instances are reported in which the onset of fibrillation seemed to be due to the administration of large doses of digitalis. Three possible causes of fibrillation are discussed:

1. Morbid structural changes in the auricle which disturb impulse conduction.
2. Alterations in the nutritional and metabolic processes in the auricular muscle.
3. Conditions of increased vagal tone.

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